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DDE reduces Medullary Bone Formation in Birds

DURING the past two decades residues of certain chlorinated hydrocarbons, especially DDT, have caused declines in the populations of various carnivorous birds in the North Temperate Zone by reducing their reproductive success¹⁻³. Contaminated birds lay eggs with abnormally thin, insufficiently calcified shells, so that there is increased egg breakage and embryonic death⁴⁻⁹. Although adult mortality, abnormal behaviour, delayed ovulation and nesting, egg eating, and failure to lay eggs also sometimes result from such contamination^{1-3,8-11}, the thin eggshell phenomenon is the chief cause of the declines in population^{3,6,8}.

Chlorinated hydrocarbons induce hepatic microsomal enzymes that hydroxylate steroids 12,13, including oestrogens, which are produced in increased amounts by the avian ovary before egg laying. In birds of both sexes, oestrogens mediate deposition of calcium in the medullaries, or hollow parts of the skeleton 14, where it serves as a labile calcium source for eggshell formation. Chlorinated hydrocarbons should therefore reduce medullary bone formation as well as eggshell calcification. We have investigated this possibility by injecting an oestrogen into male pigeons and determining whether the

ingestion of chlormated hydrocarbons by some of these birds reduces medullary bone formation, p.p. DDE1.1-dichloro-2.2-bh(p-chlorophenyl)ethylene] was used because it is the most widespread oblorinated hydrocarbon pollutant, and it has a substantial effect on eggshell thickness.

Seven groups, each of four 5 yr old male silver king pigeons, were injected twice weekly with doses of fl-oestradiol ranging from 0.05 to 0.70 mg per day during the spring of 1969. At each dose, two birds received feed containing 100 ppm of DDE dissolved in corn oil, and two control birds received feed with an equal amount of corn oil. At the end of 4 weeks, all birds were decapitated, and both femurs were removed, calcined at 675 for F h, and weighed. The average combined weight of both femurs of ten other 5 yr old male pigeons of comparable weights given neither oestradiol nor DDF was 0.793 g. This value was subtracted from all femur weights of experimental birds to give the medulfary bone weights (Table 1).

Table 1	Femur Meduil	ary Bone	Weights	
B-Oestradiol (mg/day)	Control birds		DDE birds	
0.05	0,284	0,393	0.213	0.139
0.10	0,201	-00X0	0.219	0.037
0.20	0 701	0164	0.165	0.123
0.30	0,227	0.409	0.120	0.110
0.40	0.324	0.293	0.037	0.109
0.50	0.405	0.315	0.125	0.275
0.70	0 414	0.355	0.264	0.126
Overall mean weights Expressed in %	0.283 g 190%		0.147 ջ 52.1 %	

Medullary bone formation as a function of the dose of oestradiol was inconclusive, but when the data were pooled for the two groups, medulary bone formation by the birds fed DDE was only \$2.1% as much by weight as that in control birds. The difference between the two groups was significant at the 0.01 level according to the Mann-Whitney U teres. This finding is consistent with the reduced level of circulating oestradio' found by Peakall and believed to result from induced hepatic hydroxylation?. Enzyme induction would also explain the altered behaviour, failure to lay, and late oxulation and nesting occurring among the carnivorous birds^{3,4}. Peakall and Bitman et al. have presented convincing evidence, however, that the thin-shelled eggs are caused primarily by inhibition of carbonic anhydrate, an enzyme required for shell formation in the shell-forming region of the oxiduct3.7.4. The diminished reproductive success of these birds is therefore explainable by the simultaneous occurrence of enzyme induction in the liver and enzyme inhibition in the oviduct, both resulting from these chlorinated hydrocarbon pollutants.

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